

OVERVIEW OF THE 1971 TEXAS VENEZEULAN EQUINE ENCEPHALOMYELITIS EPIZOOTIC

Richard O. Spertzel

AD 742121

The etiologic agent of Venezuelan equine encephalomyelitis (VEE) is a member of Casal's group A arboviruses. There are currently 4 major antigenic subtypes, of which the first is subdivided into 5 minor antigenic variants. The nonepizootic sylvatic subtypes occur endemically throughout major portions of Central and South America and in parts of North America¹, while major epizootics with variants 1A, 1B, and 1C have occurred in South America². The present epidemic of highly virulent subtype 1B probably had its origin in Ecuador, and was introduced into Guatemala in 1969. From there, the disease spread rapidly through El Salvador and portions of Honduras and Nicaragua. In 1970, VEE reoccurred in Honduras and spread into Costa Rica and Mexico³. Although slowed in 1970, VEE continued its inexorable spread toward the United States, and by April, 1971, was occurring near Tampico, Mexico. By early June, equine cases appeared within 35 miles of Brownsville, Texas. The first recognized encephalitic horse in South Texas was sick on 23 June. Vaccination was begun on a voluntary basis on 25 June in a 13-county area of South Texas. After confirmation of VEE by virus isolation on 9 July, vaccination was extended statewide in Texas on 13 July, and to New Mexico, Oklahoma, Arkansas, and Louisiana on 17 July. A fee-basis, Federal supported vaccination program was initiated in these 5 states. On 25 July, the vaccine area was extended to California, Arizona, Mississippi, Alabama, Georgia, and Florida, to create an ocean-to-ocean barrier. More recently, on the recommendations of a tri-agency task force, the vaccine area was extended to 8 more states: South Carolina, North Carolina, Tennessee, Kentucky, Virginia, Maryland, Delaware, New Jersey, and the District of Columbia. Missouri was invited to participate, but declined.

A State and Federal quarantine was established for Texas on 13 July; for Oklahoma, New Mexico, Arkansas, and Louisiana on 19 July; and for Mississippi on 2 August. State quarantines were established in Florida, Georgia, and Alabama. Interestingly, embargoes were placed on United States horses by Canada on 14 July. Several European countries also banned import of United States horses.

In addition to vaccination and quarantine, a mosquito-abatement program, consisting of low-volume aerial spraying of Malathion or Dibrom, was maintained along the coastal counties of Texas until 90% of the equine population was vaccinated.

Although statewide vaccination was not allowed until 13 July, suspect encephalitis cases in horses were reported in counties outside the allowable vaccine area by 10 July. Reported cases of equine illness compatible with encephalitis are shown in Fig. 1 (through 24 July) and Fig. 2 (25 July to 28 August). It should be noted that the epizootic apparently reached its maximum extent by 24 July. In

From the U.S. Army Medical Research Institute of Infectious Diseases, Frederick, Md. 21701

some of these counties, however, encephalitis in nonvaccinated horses is still occurring. Counties in which VEE virus was isolated, or specific antibody was detected in sera from nonvaccinated horses, are shown in Fig. 3. In addition, 2 border parishes of Louisiana and 2 border counties of Arkansas also reported an unusually high incidence of suspected cases of equine encephalitis. Cessation of such reports within 10 days after VEE-vaccination leads the author to suspect infection with VEE.

After the 1969 Central American epizootic and the extensive use of the live attenuated VEE vaccine in Central America and Mexico, where it appeared to be both safe and effective, the U.S. Department of Agriculture (USDA) was urged to license the vaccine for export, and for contingency use within the United States. At that time, USDA officials expressed serious reservations on the stability of the vaccine virus in this natural host, and would not authorize production of the vaccine in the United States despite authoritative recommendations to do so.

Vaccine administration to *Equidae* is characterized by a low, irregular viremia with transient fever in approximately 50% of animals.⁴ Unlike man, where 35-40% of vaccinated individuals may show some reaction to the vaccine, only 1% of horses show even a transient reaction consisting of anorexia and depression for 12-24 hours. Although no evidence of reversion to virulence was observed during serial passage of the virus by subcutaneous or intraperitoneal (IP) routes in small laboratory animals, several laboratories attempted horse-to-horse passage of the virus. Five serial passages have been attained in our laboratory and by USDA personnel with no evidence of reversion to virulence.

In addition to these back-passage studies, additional safety studies were conducted in the field. Observation of approximately 22,000 *Equidae* by USDA and/or U.S. Public Health Service personnel in 5 separate states indicated a reaction rate of less than 1%. These results were consistent with those reported by our laboratory and with empirical observations in Central America. They are in conflict with results of the limited study at another laboratory in which 3 of 6 animals showed severe depression and anorexia.⁵

In 1969 and 1970, numerous field observations attested to the efficacy of the vaccine; deaths of nonvaccinated animals were documented in herds where all vaccinees survived. These same observations have been made in Texas. A not-uncommon herd report, from an area with active encephalitic cases, follows: On 20 July, 38 working horses were vaccinated, but the owner considered it too much trouble to round up the remaining horses. In mid-August, encephalitis and death began to occur in these nonvaccinated animals. A field investigation was made on 31 August. Horses were pastured in 3 noncontiguous areas on the ranch. The 38 vaccinated horses remained healthy, while 3 of 5 unbroken geldings on the same pasture died, and one was sick at the time of investigation. In Pasture A, all 16 colts and 11 of 16 mares died; the other 5 mares were noticeably encephalitic. Similar results were seen for Pasture B.

This striking protection with one-dose immunization is consistent with the high degree of serologic conversion observed in field use of this vaccine. Of 157 paired serum samples collected during the Mexican vaccination campaign in 1970, all with preimmunization titers less than 10, 150 (96%) had HI titers greater than or equal to 20 within 30-45 days postvaccination.⁴ With this abundance of field and laboratory information, and the pressure of necessity, a provisional license for

commercial production and sale of live attenuated VEE vaccine was granted to a commercial veterinary biologics firm in July, 1971.

FIGURE 1 — Encephalitis Cases Reported On or Before 24 July 1971.

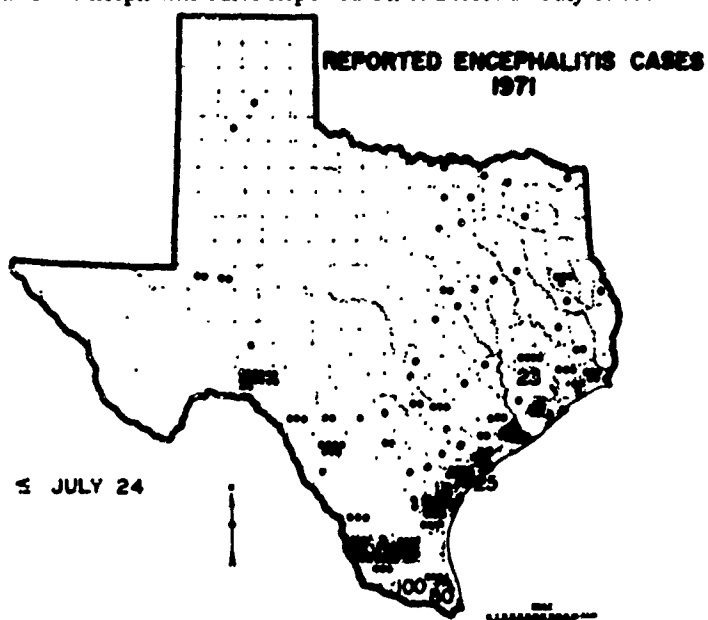


FIGURE 2 — Encephalitis Cases Reported After 24 July 1971

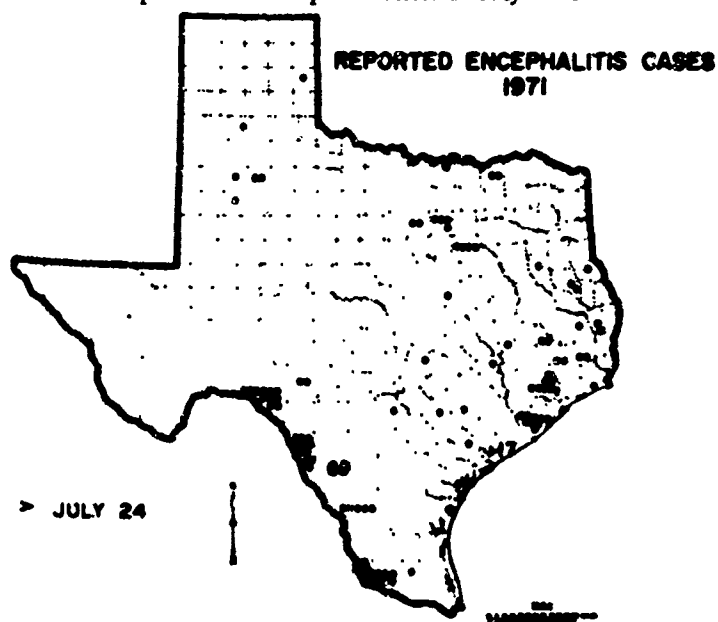
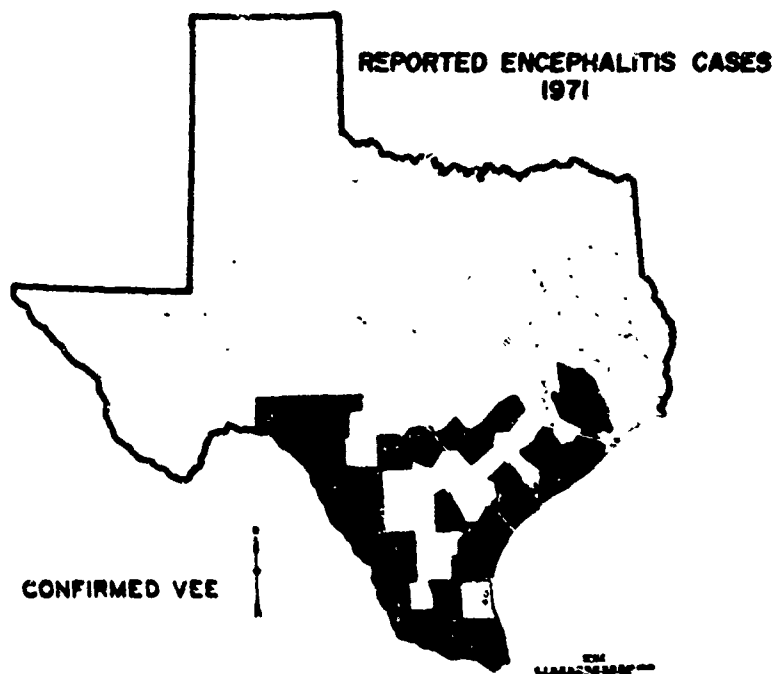


FIGURE 3 - Confirmed VEE Cases Reported, 1971



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JUSTIFICATION		
BY	DISTRIBUTION/AVAILABILITY CODES	
DATE	AVAIL. 2-3/71	SPECIAL
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